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Research Article

Cognitive Impairment and its Association with Glycemic Control among Patients with Type 2 Diabetes Mellitus

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ABSTRACT
Background: Cognitive impairment is increasingly recognized as an important complication of type 2 diabetes mellitus. Hyperglycaemia over time, recurrent hypoglycaemia, vascular comorbidities, and exposure to metabolic abnormalities, may play a role in causing memory, attention, and executive function deficits. But a lot of people who get diabetes treatment don't have cognitive function checked routinely.
Objective: To determine the frequency of cognitive impairment and evaluate its association with glycaemic control among patients with type 2 diabetes mellitus.
Methodology: A cross-sectional study was conducted in the Department of Neurology, PIMS Islamabad and Department of Neurology/Medicine Khyber Teaching Hospital, Peshawar, from January 2025 to June 2025 in hospital setting. Patients with type 2 diabetes mellitus were recruited using consecutive non-probability sampling to achieve a total of 85 patients. Demographic data, diabetes duration, treatment history, comorbidities and diabetic complications were noted. The glycated haemoglobin (HbA1c) was used to assess glycemic control, and HbA1c <7% was considered as good glycemic control, whereas HbA1c ≥7% was considered as poor glycemic control. Cognitive function was assessed with the Montreal Cognitive Assessment and a score of less than 26 was defined as cognitive impairment. Appropriate comparative tests, correlation analysis and binary logistic regression were used to assess associations.
Results: The mean age of the participants was 56.82 ± 9.74 years, and the mean duration of diabetes was 10.24 ± 6.08 years. The mean HbA1c level was 8.31 ± 1.47%, while 54 (63.5%) patients had poor glycaemic control. Cognitive impairment was identified in 38 (44.7%) patients, including 28 (32.9%) with mild and 10 (11.8%) with moderate impairment. Cognitive impairment was significantly more frequent among patients with HbA1c ≥7% than among those with HbA1c <7% (57.4% versus 22.6%, $p = 0.002$). HbA1c demonstrated a significant inverse correlation with the MoCA score ($r = -0.48$, $p < 0.001$). Poor glycaemic control, increasing age and longer duration of diabetes remained independent predictors of cognitive impairment.
Conclusion: Cognitive impairment was common among patients with type 2 diabetes mellitus and was significantly associated with poor glycaemic control. Routine cognitive screening, particularly among older patients and those with longstanding or poorly controlled diabetes, may support early detection and safer individualized management.

Keywords: Cognitive Impairment, Type 2 Diabetes Mellitus, Glycaemic Control, Hba1c, Montreal Cognitive Assessment, Hyperglycaemia.

INTRODUCTION

Diabetes mellitus is a chronic metabolic disorder that is defined by hyperglycaemia for more than a couple of weeks, due to a defect in insulin secretion, insulin action, or both. Most cases are attributed to type 2 diabetes mellitus, which is a major public health problem worldwide. Some 830 million people in the world suffer from diabetes, and many of them live in low-resource countries. The disorder is linked to a progressive damage to blood vessels, nerves, kidneys, eyes and the cardiovascular system, contributing to a significant amount of disability, early death and health care costs [1-3].

While vascular, renal, retinal and neurological complications of diabetes are well documented, the impact of diabetes on cognitive function has been less studied in routine clinical practice. Diabetic patients have a higher risk of cognitive dysfunction, MCI and dementia than non-diabetic patients. Symptoms of cognitive decline can start with mild problems in memory, attention, information-processing speed and executive functioning, and may progress to more severe memory impairments that impact a person's ability to engage in independent daily living. The clinical guidelines for the management of diabetes today agree that cognitive dysfunction can negatively impact compliance, self-monitoring of blood glucose, meal planning, self-insulinization and hypoglycaemia awareness [4-6].

Diabetes-related cognitive impairment is a complex, multi-factorial disease process. Chronic hyperglycaemia may contribute to cause oxidative stress, advanced glycation end-product formation, endothelial dysfunction, cerebral microvascular injury and neuroinflammation. Insulin resistance in the CNS can affect synaptic plasticity and neuronal survival; repeated hypoglycaemia can lead to the brain being deprived of its main energy source and cause neuronal injury. Other factors such as hypertension, dyslipidaemia, obesity, diabetic microvascular complications and ageing can make people more susceptible to cognitive decline. A few studies have recently found that high HbA1c and low educational levels, plus advanced age and duration of diabetes, are all key factors for the presence of mild cognitive impairment (MCI) among people with type 2 diabetes [7-9].

Glycaemic control may be particularly important in determining cognitive outcomes. HbA1C is a good indicator of AVERAGE blood glucose level over the past 2-3 months and is a routine blood testing required for long term glycaemic control. There are links between higher HbA1c, fasting glucose and PPG levels with reduced cognitive function, suggesting that long term hyperglycaemia could negatively impact on cognitive function. But, the relationship can also be two-way as patients with cognitive impairment might struggle to adhere to multiple treatment regimens and may find it challenging to control glucose. Another risk is hypoglycaemia, which can occur with intensified treatment of the patient, especially the elderly with reduced mental abilities [10-12] .

If cognitive impairment is identified early, a more simple treatment regimen can be used, caregivers can be engaged in treatment, and glycaemic targets can be individualized. The Montreal Cognitive Assessment is a quick multidomain screening tool to assess executive function, visuospatial ability, memory, attention, language, abstraction and orientation. It has been shown to be highly sensitive in identifying mild cognitive impairment in people with type 2 diabetes, but its findings need to be interpreted based on educational, linguistic and cultural background [13].

Despite the clinical importance of diabetes-related cognitive dysfunction, local evidence regarding its frequency and relationship with glycaemic control remains limited. Cognitive assessment is also not commonly incorporated into routine diabetes follow-up in many healthcare settings. Therefore, the present study was conducted to determine the frequency of cognitive impairment and assess its association with glycaemic control among patients with type 2 diabetes mellitus presenting to Department of Neurology, PIMS Islamabad and Department of Neurology/Medicine Khyber Teaching Hospital, Peshawar. It was hypothesized that patients with poor glycaemic control would have a higher frequency of cognitive impairment and lower cognitive-assessment scores than patients with good glycaemic control.

METHODOLOGY

This hospital-based cross-sectional study was conducted in the Department of Neurology, PIMS Islamabad and Department of Neurology/Medicine Khyber Teaching Hospital, Peshawar, from January 2025 to June 2025. The study aimed to determine the frequency of cognitive impairment and assess its association with glycaemic control among patients diagnosed with type 2 diabetes mellitus.

Written informed consent was obtained from all participants after explaining the purpose, procedures, potential benefits and confidentiality measures of the study. Participation was voluntary, and patients were assured that refusal to participate would not affect their routine medical care.

The number of patients who had type 2 diabetes mellitus was 85, they were selected consecutively and non-probability sampling was used. Adult (age 30 and over) males and females with a documented history of Type 2 Diabetes Mellitus (T2DM) for at least one year were eligible. Patients who came to the outpatient department or were admitted to the medical wards over the study period were evaluated for eligibility. Patients with a prior neurologic and/or cognitive disorder, dementia, stroke, epilepsy, severe psychiatric disorder, head trauma or infection of the central nervous system that would significantly impact cognitive testing, impaired hearing and/or vision that would interfere with cognitive testing and those taking medications known to significantly affect cognition were excluded. The following exclusion criteria were applied: acute ill, haemodynamically unstable, patients who were unable to communicate and patients who were unable to provide informed consent.

Once enrolled, demographic and clinical data were taken with a structured data collection proforma. The recorded variables were: age, sex, educational level, residence, smoking history, body mass index, duration of diabetes, type of antidiabetic treatment, history of hypoglycaemic episodes, hypertension, and dyslipidaemia and diabetes-related complications. The height was measured by a stadiometer and weight using calibrated weighing scale. BMI was defined as the ratio of weight (kg) to height (m^2). Diabetic neuropathy, retinopathy and nephropathy information was collected through clinical exam and available medical records. Diabetes duration was defined as the number of years between the date of diagnosis and date of enrolment, once a diagnosis has been confirmed.

Venous blood samples were used to test glycated haemoglobin to check glycaemic control. Blood was drawn under sterile conditions and the analysis was performed in the hospital laboratory by standardised methods. HbA1c values were categorized as good glycaemic control (HbA1c < 7%) or poor glycaemic control (HbA1c ≥ 7%). Fasting blood glucose was also taken after at least 8 hours of night time fasting. The Montreal Cognitive Assessment (MCA) was used to assess cognitive function in a quiet setting and was administered by a trained investigator. Visuospatial and executive function, naming, attention, language, abstraction, delayed recall and orientation were evaluated. A score less than 26 on the total MoCA was regarded as indicative of cognitive impairment. For those who completed 12 years or less of formal education, an additional point was awarded.

The data were coded and analyzed using IBM SPSS Statistics 26. Continuous variables (age, BMI, diabetes duration, HbA1c, FBG, MoCA) were expressed as mean ± SD or median (IQR) as appropriate. Frequencies and percentages were used to present the categorical variables. Independent-samples t-test or Mann–Whitney U test was used to compare continuous variables between the patients with and without cognitive impairment. Association between categorical variables was tested using either the chi-square test or Fisher's exact test. Pearson or Spearman correlation analysis was performed to determine the relationship of HbA1c, age and duration of diabetes with the MoCA score. After controlling for the potential confounding variables, binary logistic regression analysis was performed to determine independent predictors of cognitive impairment. Odds ratios with 95% confidence intervals were reported and a p value < 0.05 was deemed statistically significant.

RESULTS

The total number of patients included in the study was 85 with T2DM. The average age of the subjects was 56.82 ± 9.74 years, and ranged from 38 to 76 years. Most patients were aged 51–60 years (35.3%), followed by those older than 60 years (30.6%). There were 45 (52.9%) males and 40 (47.1%) females. The mean duration of diabetes was 10.24 ± 6.08 years, while the mean body mass index was 27.63 ± 4.18 kg/m^2. The majority of the participants had hypertension (48, 56.5%) while the majority reported having dyslipidaemia (37, 43.5%). Forty-six (54.1%) patients were taking oral hypoglycaemic agents, 21 (24.7%) were taking insulin alone and 18 (21.2%) were taking a combination of both.

Table 1. Sociodemographic and Clinical Characteristics of Participants (N = 85)

Variable	Frequency (%) or Mean ± SD
Age, years	56.82 ± 9.74
30–40 years	6 (7.1)
41–50 years	23 (27.1)
51–60 years	30 (35.3)
>60 years	26 (30.6)
Male	45 (52.9)
Female	40 (47.1)
Diabetes duration, years	10.24 ± 6.08
Diabetes duration <5 years	18 (21.2)
Diabetes duration 5–10 years	29 (34.1)
Diabetes duration >10 years	38 (44.7)
Body mass index, kg/m²	27.63 ± 4.18
Hypertension	48 (56.5)
Dyslipidaemia	37 (43.5)
Current smoker	17 (20.0)
Oral hypoglycaemic agents	46 (54.1)
Insulin therapy	21 (24.7)
Combined therapy	18 (21.2)
History of hypoglycaemia	24 (28.2)
Diabetic neuropathy	29 (34.1)
Diabetic retinopathy	20 (23.5)
Diabetic nephropathy	14 (16.5)

The average HbA1C level of the subjects in the study was 8.31 ± 1.47%. Thirty-one (36.5%) patients had good glycaemic control (HbA1c < 7%) compared with 54 (63.5%) with poor glycaemic control. The mean fasting blood glucose level was 161.47 ± 46.28 mg/dL. The Montreal Cognitive Assessment was used to assess cognitive function. The mean MoCA score was 23.42 ± 3.68.

Table 2. Glycaemic Profile and Cognitive Assessment Findings

Variable	Result
HbA1c, %	8.31 ± 1.47
HbA1c <7%	31 (36.5)
HbA1c ≥7%	54 (63.5)
Fasting blood glucose, mg/dL	161.47 ± 46.28
MoCA score	23.42 ± 3.68
Normal cognitive function	47 (55.3)
Cognitive impairment	38 (44.7)
Mild cognitive impairment	28 (32.9)
Moderate cognitive impairment	10 (11.8)
Severe cognitive impairment	0 (0.0)

Altogether, 44.7% (38/85) of participants was determined as having cognitive impairment. Of those who were affected, 28 (32.9% of the entire sample) had mild cognitive impairment, and 10 (11.8%) had moderate cognitive impairment. No patient was considered as severe cognitive impairment. Poor glycaemic control was significantly associated with cognitive impairment. Of the 54 patients with HbA1c ≥7%, 31 (57.4%) had cognitive impairment, compared with 7 of the 31 patients (22.6%) with HbA1c <7%. This difference was significantly different ($\chi^2 = 9.66$, $p = 0.002$).

Table 3. Association between Glycaemic Control and Cognitive Impairment

Glycaemic control	Normal cognition n (%)	Cognitive impairment n (%)	Total	p-value
HbA1c <7%	24 (77.4)	7 (22.6)	31	

HbA1c ≥7%	23 (42.6)	31 (57.4)	54	**0.002**
Total	47 (55.3)	38 (44.7)	85	

Patients with cognitive impairment were older than those with normal cognitive function (mean age: 60.34 ± 8.27 years vs. 53.98 ± 9.96 years, respectively, p = 0.002). Patients with cognitive impairment also had longer duration of diabetes compared with those with normal cognitive function (13.21 ± 5.89 years versus 7.84 ± 5.14 years, p < 0.001). The cognitively impaired group had a significantly higher mean HbA1c level than the group with normal cognitive function (9.02 ± 1.31% compared to 7.74 ± 1.34%, p < 0.001). Likewise, FPG was significantly elevated in patients with cognitive impairment. Patients with cognitive impairment also had a higher prevalence of hypertension, a history of hypoglycaemia and diabetic neuropathy and diabetic retinopathy.

Table 4. Comparison of Patients with and Without Cognitive Impairment

Variable	Normal cognition (n = 47)	Cognitive impairment (n = 38)	p-value
Age, years	53.98 ± 9.96	60.34 ± 8.27	**0.002**
Male sex	27 (57.4)	18 (47.4)	0.357
Diabetes duration, years	7.84 ± 5.14	13.21 ± 5.89	**<0.001**
HbA1c, %	7.74 ± 1.34	9.02 ± 1.31	**<0.001**
Fasting blood glucose, mg/dL	146.26 ± 38.72	180.29 ± 48.61	**0.001**
BMI, kg/m²	27.18 ± 4.03	28.19 ± 4.34	0.270
Hypertension	21 (44.7)	27 (71.1)	**0.015**
History of hypoglycaemia	8 (17.0)	16 (42.1)	**0.011**
Diabetic neuropathy	10 (21.3)	19 (50.0)	**0.005**
Diabetic retinopathy	6 (12.8)	14 (36.8)	**0.009**
Diabetic nephropathy	5 (10.6)	9 (23.7)	0.105

There was a statistically significant negative correlation between HbA1C level and MoCA score (r = −0.48, p < 0.001) by Pearson correlation analysis. This suggested a negative correlation between HbA1c level and cognitive ability. MoCA score was also negatively correlated with duration of diabetes (r = −0.41, p < 0.001), while there was a moderate negative correlation between age and cognitive performance (r = −0.36, p = 0.001).

Table 5. Correlation of Clinical Variables with Moca Score

Variable	Correlation coefficient (r)	p-value
HbA1c level	−0.48	**<0.001**
Duration of diabetes	−0.41	**<0.001**
Age	−0.36	**0.001**
Fasting blood glucose	−0.33	**0.002**
Body mass index	−0.14	0.206

Categorical variables that had clinically important and/or statistically significant relationships with the response variable in the univariate analyses were included in a binary logistic regression model. Poor glycaemic control was still independently associated with cognitive impairment after adjustment for age, sex, educational level, length of diabetes, hypertension, hypoglycaemia and diabetic complications. Approximately 3.42 times higher odds of cognitive impairment were found in the patients with HbA1c ≥7% as compared to those with HbA1c <7% (3.42, 1.19–9.81, p = 0.022). Other independent predictors were increasing age and increasing duration of diabetes. For every extra year in age, there was a 7% increase in risk of cognitive impairment; for every 10 years of diabetes, there was a 9% increase in risk of cognitive impairment. There was also an association with a history of recurrent hypoglycaemia and an increased likelihood of cognitive impairment, but with a wide confidence interval.

Table 6. Binary Logistic Regression Analysis of Factors Associated with Cognitive Impairment

Predictor	Adjusted odds ratio	95% confidence interval	p-value
HbA1c ≥7%	3.42	1.19–9.81	**0.022**
Age, per one-year increase	1.07	1.01–1.14	**0.025**
Diabetes duration, per one-year increase	1.09	1.01–1.18	**0.031**
Hypertension	1.86	0.68–5.11	0.228
History of hypoglycaemia	2.74	0.91–8.24	0.073
Diabetic neuropathy	2.16	0.76–6.15	0.150
Female sex	1.24	0.46–3.31	0.672

Overall, cognitive impairment affected nearly half of the patients with type 2 diabetes mellitus. Poor glycaemic control, advanced age and longer duration of diabetes were the principal factors associated with impaired cognitive performance.

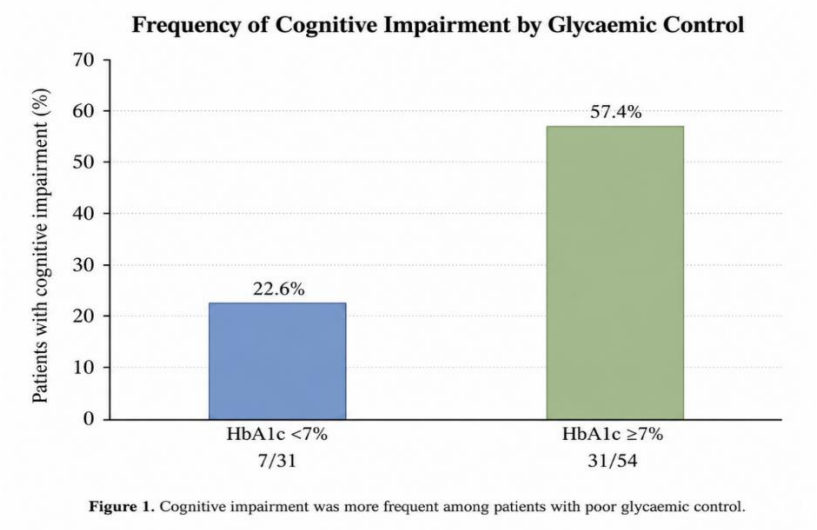

Figure 1. Comparison of the Frequency of Cognitive Impairment among Patients with Good Glycaemic Control (Hba1c <7%) and Poor Glycaemic Control (Hba1c ≥7%). Cognitive Impairment was Significantly More Frequent in Patients with Poor Glycaemic Control.

DISCUSSION

The present study demonstrated that cognitive impairment was common among patients with type 2 diabetes mellitus, affecting 44.7% of the study population. Mild cognitive impairment was identified in 32.9% of participants, whereas 11.8% had moderate impairment. These findings indicate that cognitive dysfunction may represent an important but frequently under-recognized complication of diabetes. A recent study evaluating patients with type 2 diabetes reported mild cognitive impairment in 56% of participants, with a mean MoCA score of 25.14 ± 1.63 [14]. Another population-based investigation found that individuals with diabetes had a significantly higher prevalence of mild cognitive impairment than those without diabetes, 19.9% versus 14.8%, and also experienced a higher incidence of cognitive impairment during long-term follow-up [15]. Differences in reported frequencies may be related to variations in participants' age, educational level, duration of diabetes, cognitive assessment method, HbA1c status and burden of vascular complications.

One of the primary conclusions of the current study was that there was a strong association between poor glycaemic control and cognitive impairment. Patients with HbA1C ≥7% had significantly more cognitive impairment (57.4%) than those with HbA1C <7% (22.6%) ($p = 0.002$). Additionally, the mean HbA1c was significantly elevated in patients with cognitive impairment as compared to patients with normal cognition; MoCA score was moderately inversely correlated with HbA1c. These results are congruent with those showing that long-term hyperglycaemia and glycaemic variability are linked to worse cognitive function in T2DM patients [16]. Linear associations between cognitive impairment and fasting glucose and postprandial glucose and HbA1c levels were

also revealed in a 2024 meta-analysis [17]. The chronic hyperglycaemic state can lead to cognitive dysfunction, via mechanisms of oxidative stress, advanced glycation end products, endothelial dysfunction, cerebral microvascular disease, neuroinflammation and impaired neuronal insulin signalling. However, the current study was cross-sectional, so it would be inappropriate to conclude that increased HbA1c was the direct cause of cognitive dysfunction.

Increasing age and longer duration of diabetes were also significantly associated with cognitive impairment. The patients with impaired cognition were older and had a longer duration of diabetes than patients with normal cognition. Both age and diabetes duration were independent predictors of cognitive impairment in the adjusted analysis. The findings are consistent with a recent meta-analysis which showed that advanced age, diabetes duration, HbA1C and lower educational levels were significant prognostic factors for mild cognitive impairment in T2DM patients [18]. Exposure to metabolic disturbances for a longer period of time may result in greater cumulative effects of hyperglycaemia, insulin resistance, vascular disease and repeated glycaemic changes on cerebral structure and function. Cognitive reserve may also be depleted and the risk for vascular and neurodegenerative changes may be accentuated with ageing. It is therefore recommended that patients with diabetes receive cognitive screening every time they come to the clinic for their diabetes maintenance, particularly those who are older or have had diabetes for a long time.

In the present study, hypertension, previous hypoglycaemic episodes, diabetic neuropathy and diabetic retinopathy were also more common in the patients with cognitive impairment. Hypertension can speed up the progression of cerebral small vessel disease, while retinopathy, neuropathy and nephropathy may be markers of generalized microvascular complications of long-term diabetes. This is supported by recent data that diabetic microvascular complications are also a significant factor in the burden of cognitive impairment, and that there are important relationships between nephropathy and other vascular complications with cognitive decline [19]. Clinically, the link with hypoglycaemia is of importance as it could be suggested that cognitive impairment and hypoglycaemia may be bidirectional. Recurrent hypoglycaemia can have a negative impact on cognitive function, which, in turn, can affect a patient's capacity to adhere to treatment, to self-manage insulin dosage and to identify signs and symptoms and respond appropriately when blood glucose falls. The current standard of care for diabetes, then, suggests an annual cognitive evaluation of older people with diabetes and adjustment or simplification of the treatment plan if cognitive issues hinder safe diabetes self-care [20].

The results of the study have implications for diabetes care in Khyber Teaching hospital and other health care institutes. The use of a short cognitive screening test like the MoCA in assessing high-risk patients may facilitate earlier identification of cognitive problems and facilitate adjustments to treatment plans. The MoCA has been shown to be valid for cognitive impairment in persons with type 2 diabetes, but it should be interpreted based on language, education and cultural context [8]. Patients with cognitive impairment may need to have their medication regimens simplified, written instructions, involvement of their caregiver(s) and avoidance of complex insulin regimen, and use of individualized glycaemic targets that consider the risks of hyperglycaemia and hypoglycaemia. The present study's design was limited to a single centre, a relatively small sample size, and the assessment being based on a screening tool rather than on full neuropsychological testing. Possible confounding from depression, social, physical activity, sleep disorders, and medication use also may have affected cognition. Long-term larger multicentre prospective studies are suggested to clarify the long-term effect of better and more stable glucose control on cognition.

CONCLUSION

Cognitive impairment was frequent among patients with type 2 diabetes mellitus, affecting nearly half of the study population. Poor glycaemic control was strongly associated with impaired cognitive performance, and patients with HbA1c ≥7% had a substantially higher frequency of cognitive impairment than those with better glycaemic control. Advanced age and longer duration of diabetes were independent predictors, while hypertension, hypoglycaemic episodes and diabetic microvascular complications were also associated with cognitive impairment. Routine cognitive screening, particularly among older patients, individuals with longstanding diabetes and those with persistently elevated HbA1c, may facilitate early detection and safer

individualized diabetes management. Prospective multicentre studies are required to clarify the temporal relationship between glycaemic control and cognitive decline.

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